The Egyptian Journal of Hospital Medicine (October 2025) Vol. 101, Page 5371-5377

Assessment of Left Ventricular Function in Patients with Ventricular Septal Defect Using Speckle Tracking Echocardiography

[*1]Kholoud Ata Ismael*, [1]Dina Youssef Nassar, [1]Safaa Abo-Alfadl Mohammed, [2]Rania Abu Shokka

[1]Department of Cardiology, Faculty of Medicine for Girls, Al-Azhar University

[2]Department of Cardiology, National Heart Institution, Egypt

***Corresponding author: Kholoud Ata Ismael Ali**, **Mobile:** 01033487056,

ORCID: 0009-0009-2543-532X , **Email:** kholodata881@gmail.com

ABSTRACT

Background: Up to 40% of all cardiac abnormalities are caused by ventricular septal defects (VSD), the most prevalent congenital heart defect (CHD). Hemodynamic overload in children /with VSDs leads to myocardial damage and eventual heart failure. Conventional echocardiography cannot identify cardiac injury in its early stages. A more precise method for the early detection of cardiovascular disorders is speckle tracking echocardiography (STE), which was just introduced.

Aim of the work: This study aimed to assess the left ventricular function in patients with isolated VSD using speckle-tracking echocardiography (STE).

Patients and methods: The study was conducted on 30 patients with isolated VSD, with a mean age 9.02 ± 4.90 years, in addition to 20 healthy age- and sex-matched control group. The VSD group was further stratified according to age .into two subgroups: Group A included 14 patients aged 2-8 years. Group B involved 16 patients aged 9-17 years Echocardiography evaluation included M-mode, 2D, color Doppler, TDI and STE.

Results: In our study; LVGLS was significantly lower in patients with VSD (-20.43 ± 1.92) compared to the control group [(-21.61±1.27), p value<0.05]. Subgroup analysis revealed no significant difference regarding LV- GLS in group A (-21.07 ± 1.85) compared to their controls (-22.06 ± 1.32) (P=0.182). On the other hand, group B had significantly lower values of LV-GLS (-19.33 ± 1.85) compared to the normal group (-21.24 ± 1.16) (p = 0.039). LVGLS correlated negatively with the left atrial diameter (r = -0.38, P = 0.029), left ventricular dimensions (r = -0.416, P = 0.024), VSD size (r =-0.392, P=0.017), Qp/Qs ratio (r =-0.480, P=0.007) and age "years" (r=-0.513, P=0.004).

Conclusion: STE is a sensitive and valuable tool for the early detection and prediction of subclinical LV dysfunction in patients with VSD, even when conventional echocardiographic parameters appear normal.

Keywords: Speckle tracking echocardiography, Left ventricular function, Isolated VSD.

INTRODUCTION

Congenital heart diseases (CHDs) affect approximately 8–12 infants per 1,000 live births. While many CHDs are identified antenatally or during early childhood, diagnosis may occasionally be delayed until adulthood [1]. VSD accounts for around 40% of all CHDs. Isolated VSDs represent more than 20% of these cases [2]. VSD are classified based on their anatomic relationship to the septum into inlet, trabecular (muscular), outlet (supracristal) and membranous types. The most prevalent subtype is the perimembranous VSD, which constitutes approximately 80% of all VSDs [3]. Less common types include muscular, inlet, and outlet VSDs [4]**.** A left-to-right shunt across a VSD results in volume overload and dilation of the left atrium and left ventricle, which may eventually lead to left ventricular (LV) dysfunction [5]. The hemodynamic impact and the degree of shunting depend on the defect size, the pressure gradient between the right and left ventricles and the pulmonary vascular resistance [6].

Echocardiography is the primary imaging modality for evaluating CHDs. It is non-invasive, widely available, cost-effective and provides detailed structural information. It is considered the "workhorse" of CHDs assessment and follow-up [1].

The left ventricular ejection fraction (LVEF) remains a strong predictor of mortality and guides decisions regarding device therapy and surgical interventions. It is highly load-dependent and significantly influenced by the operator's experience and technical expertise [7,8]. In recent years, STE has emerged as the modality of choice for the assessment of myocardial strain and deformation. Unlike conventional echocardiographic techniques, STE is angle-independent and allows for early detection of subclinical myocardial dysfunction [9].

PATIENTS AND METHODS

This case-control study was conducted on 50 participants: 30 patients with unrepaired isolated restrictive ventricular septal defects (VSD), aged between 2 and 17 years, and 20 age- and sex-matched healthy controls. All participants were recruited from the Cardiology Outpatient Clinics at Al-Zahraa University Hospital and the National Heart Institute, after obtaining verbal consent. The study period extended from December 2023 to January 2025.

Exclusion criteria: Patients with congenital heart diseases other than VSD, large VSD, severe systemic illness, concomitant cardiac diseases (valvular, ischemic or myopathic), or serious arrhythmias

All studied cases were subjected to the following assessments:

(I) Thorough medical history and physical examination: Demographic information: Sex, age, body surface area (BSA), height and weight. The

Received: 24/05/2025
Accepted: 26/07/2025

Mosteller formula was used to determine BSA. Shortness of breath, fast breathing, palpitations, perspiration, intolerance to activity and poor nutrition are all signs of volume overload. The child (if old enough) or caregiver reports were used to identify these symptoms. **Physical examination:** For syndromic characteristics or other congenital abnormalities. Assessing for pericardial bulge, surgical scars, dextrocardia, left parasternal thrill, hyperdynamic apex and auscultation of heart sounds were all part of the cardiac examination. Systolic murmurs, presence of S4 or gallop rhythm, exaggerated second heart sound (S2) and indications of pump failure (pulmonary or systemic venous congestion) were among the other abnormalities assessed.

(II) Twelve-lead surface ECG: A standard 12-lead ECG was performed for all cases and thoroughly evaluated for heart rate, rhythm, QRS axis and duration, as well as findings suggestive of right ventricular enlargement (RVE) or the presence of right bundle branch block (RBBB).

(III) Transthoracic Echocardiography (TTE): Children under 3 years of age were sedated with chloral hydrate [10]**.** Transthoracic echocardiographic Doppler studies were performed for all participants using the VIVID E9 GE system, equipped with tissue Doppler and speckle tracking capabilities, connected to the EchoPAC workstation (version 113).

The **left ventricular functions** were assessed using a variety of echocardiographic modalities, including M-mode, 2D conventional Doppler, tissue Doppler imaging (TDI) and (STE), along with comprehensive evaluation of the VSD.

The following parameters were assessed: ***VSD Assessment:*** Multiple-view visualization of the VSD. The site and quantity of VSDs, the greatest pressure gradient across the defect, the existence of chamber dilatation, pulmonary hypertension, and related cardiac abnormalities were among the evaluation parameters.

VSD size was determined by measuring the largest diameter using color flow (CF) Doppler. Shunt direction was assessed using color flow mapping, and shunt fraction (Qp/Qs) was calculated using the following measurements:

Left ventricular outflow tract (LVOT) diameter from the parasternal long axis view & LVOT velocity time integral (VTI) from the apical 5-chamber view.

Right ventricular outflow tract diameter (RVOT) and velocity time integral (RVOT-VTI) from the parasternal, **viewaxis shorts** .

(Qp/Qs) = [(pulmonary artery diameter) 2 × pulmonary artery velocity time integral] / [(left ventricular outflow tract diameter) 2 × left ventricular outflow tract velocity time integral] was the noninvasive formula used to get the Qp/Qs ratio [11]**.**

Left ventricular Echo-Doppler parameters:

Conventional parameters: From 2D echo-guided M-mode imaging in the parasternal long-axis or short-axis views, the following measurements were obtained: Left ventricular end-diastolic diameter (LVEDD) and end-systolic diameter (LVESD). Left atrial (LA) dimensions. Left ventricular percent fractional shortening (PFS): PFS was calculated as: [100 * (LVEDD - LVESD) / LVEDD]. A normal PFS is > 25%. M-mode-derived left ventricular ejection fraction (EF): LVEF was calculated using the Teichholz method and measured automatically by the machine software. Normal ejection fraction is 53-73% [52-72% for male patient & 54-74% for female] [12].

- **2D echo biplane LVEF:** It is measured by tracing of the blood-tissue interface in the apical four-chamber and the apical two-chamber views. Then ejection fraction is calculated automatically from EDV and ESV estimates, using the following modified Simpson's formula:

EF% = 100 * **(LVEDV - LVESV) / (LVEDV)**

Doppler transmitral flow velocities for pulsed wave Doppler (PWD): In order to measure the mitral peak early diastolic velocity (MV-E vel), late diastolic velocity (MV-A vel) wave, MV-E/A ratio and mitral valve deceleration time (MV-DT), Doppler mitral inflow velocities were recorded from the apical four-chamber view pulse wave by placing sample volume at the tips of the mitral valve (MV) [13].

Left ventricular parameters obtained by Newer Echo Modalities:

Tissue Doppler Imaging (TDI) Measures: Pulsed-wave TDI (1–5 MHz) was utilized with low filter settings (50 Hz) and optimally adjusted gains to ensure high-quality velocity signals. Using the smallest optimum gain and spectral Doppler signal filters tuned to attain a Nyquist limit of 15–20 cm/s, a 4-mm sample volume was used. To enable a frame rate higher than 90 frames per second, the image sector width was reduced as much as feasible. To prevent aliasing inside the image, particular attention was given to the color Doppler velocity range settings [14].

In the apical 4-chamber view, the sample volume was placed in the ventricular myocardium next to the lateral and septal mitral annulus. Measurements were then averaged from these two locations.

The following TDI parameters were used to evaluate LV function: MV-Sa, or mitral annular systolic velocity. MV-Ea, or peak early diastolic velocity. MV-Aa, or peak late diastolic velocity. The ratio of the mitral inflow E-wave (as determined by pulsed-wave Doppler) to MV-Ea is known as the MV E/Ea ratio.

(STE) for LV Deformation Assessment:

Left ventricular global longitudinal strain (LV-GLS) was assessed using **automated functional imaging (AFI)** for 2D speckle-tracking analysis. A high-quality ECG tracing was utilized, with the **QRS complex** marking the beginning of systole and the **T wave** marking its end.

Image acquisition was performed with attention to the following criteria: Appropriate depth to include the

mitral annulus in diastole, ensuring clear visualization of the leaflet insertion points at the annulus. Appropriate sector acquisition, including: a. Complete visualization of the apex in diastole. b. Partial inclusion of the right ventricle (RV) to ensure full capture of the interventricular septum. c. Clear definition of the epicardial borders of the anterior and lateral walls, with a frame rate between 40–70 frames/s. Appropriate endocardial definition for accurate assessment of left ventricular volumes and ejection fraction (EF). Use of slight over-gain to facilitate endocardial tracking. Avoidance of foreshortening, which can result in overestimation of apical strain. LV-GLS was calculated using AFI. The endocardial border was automatically traced by the software from three standard apical views: Apical long-axis, apical 4-chamber, and apical 2-chamber. Manual adjustments were made when necessary to optimize tracking. In healthy children, mean LV GLS was -20.2% [15].

Study Groups:

A. Main groups

VSD Group: Patients diagnosed with ventricular septal defect.

Control Group: Age- and sex-matched healthy subjects.

B. Subgroups within the VSD cohort

Group A: Fourteen patients aged 2–8 years.

Group B: Sixteen patients aged 9–17 years.

Ethical consideration: The study protocol was reviewed and approved by The Medical Ethics Committee of Al-Azhar Faculty of Medicine for Girls (AFMG) (Study ID: 2217). Written informed consents were obtained from all participants. This study was conducted in accordance with the World Medical Association's Code of Ethics (Declaration of Helsinki) for research involving humans.

Statistical analysis

The statistical software for social sciences, version 23.0 (SPSS Inc., Chicago, Illinois, USA), was used to evaluate the recorded data. In the case of parametric (normal) distribution, the quantitative data were displayed as mean ± standard deviation and ranges, whereas non-parametric (non-normally distributed) variables were displayed as median with inter-quartile range (IQR). Qualitative variables were also displayed as percentages and numbers. Using the Shapiro-Wilk and Kolmogorov-Smirnov tests, the data were examined for normality. $P \leq 0.05$ was deemed significant.

RESULTS

This study included a total of 50 participants. Among them, 30 patients were diagnosed with isolated ventricular septal defect (VSD group), while 20 age- and sex-matched healthy individuals served as the control group. The studied VSD patients included 10 males (33.3%) and 20 females (66.7%), while the control group included 9 males (45.0%) and 11 females (55.0%). The mean age of the patient group (VSD) was 9.02 ± 4.90, while the mean age of the control group was 9.25 ± 4.55. (p>0.05) (Table 1).

Table (1): Characteristics of the included participants

Parameter	VSD group (n=30)	Normal group (n=20)	p-value
Age (years)	9.02 ± 4.90	9.25 ± 4.55	>0.05
Sex			
Male	10 (33.3%)	9 (45.0%)	>0.05
Female	20 (66.7%)	11 (55.0%)	

The majority of ventricular septal defects (VSDs) were perimembranous (80%), with muscular defects accounting for the remaining (20%). The mean VSD size was 4.00 ± 1.11 mm. The mean pressure gradient across the VSD was 92.10 ± 10.52 mmHg. The mean Qp/Qs ratio was 1.63 ± 0.21, indicating a moderate left-to-right shunt volume, with values approaching 2 in some patients (Table 2).

Table (2): VSD type and shunt distribution among patient group

Perimembranous VSD	24 (80.0%)
Muscular VSD	6 (20%)
VSD size	4.00 ± 1.11 mm
PG across VSD	92.10 ± 10.52 mmHg
RVOT VTI	22.39 ± 4.31 cm
RVOT diameter	19.00 ± 3.32 cm
Systemic VTI	19.81 ± 3.23 cm
LVOT diameter	15.87 ± 2.91 cm
Qp /Qs ratio	1.63 ± 0.21

Among the VSD group, 40% of patients were symptomatic. All symptomatic patients reported dyspnea classified as NYHA class II (P = 0.001). Additionally, 10 patients (33.3%) experienced fatigue, and 5 patients (16.7%) reported palpitations (Table 3).

Table (3): Clinical presentation of VSD group

	VSD group (n=30)
Symptomatic	12 (40.0%)
Asymptomatic	18 (60.0%)
Dyspnea	12 (40,0%)
Palpitation	5 (16.7%)
Easy fatigability	10 (33.3%)

When compared to the control group, the VSD group's left ventricular end-diastolic diameter (LVEDD) and left atrial diameter (LAD), which were substantially larger (p = 0.030 and p = 0.009, respectively). On the other hand, there was no discernible difference between the two groups in terms of LVESV, LLVEDV, EF%, or LVESD (Table 4).

Table (2): Comparison of LV conventional M-mode and 2D Echocardiographic parameters between VSD and control groups

	VSD group (n=30)	Normal group (n=20)	Test value	p-value
LVEDD (mm)	39.57 ± 5.98	36.05 ± 4.54	4.987	0.03*
LVESD (mm)	25.10 ± 4.26	23.70 ± 4.64	1.208	0.277
FS (%)	36.53 ± 3.68	37.45 ± 2.76	0.899	0.348
EF (%)	66.73 ± 5.09	68.20 ± 3.56	1.25	0.269
LAD (mm)	29.93 ± 4.39	26.60 ± 4.02	7.402	0.009*
LVEDV	52.83±19.94	44.65±15.97	1.355	0.131
LVESV	19.70±8.78	16.30±6.37	1.216	0.143

Regarding mitral inflow characteristics, such as MV-E velocity, MV-A velocity, MV-E/A ratio, and MV deceleration time (MV-EDT), no statistically significant changes were found between the two groups (Table 5).

Table (3): Comparison of LV conventional Doppler flow parameters between VSD and control groups

	VSD group (n=30)	Normal group (n=20)	Test value	p-value
MV-E velocity(m/s)	0.97 ± 0.11	0.98 ± 0.15	0.005	0.942
MV A velocity(m/s)	0.60 ± 0.11	0.56 ± 0.13	1.618	0.209
MV-DT(s)	130.23 ± 19.46	132.30 ±16.82	0.15	0.7
E/A ratio	1.71 ± 0.34	1.83 ± 0.42	1.208	0.277

When compared to the control group, the VSD group's mitral annular early diastolic velocity (Av. Ea) was considerably lower (p = 0.002). Furthermore, both metrics stayed within the typical reference range, but the E/Ea ratio was noticeably greater in the VSD group (p = 0.048). In contrast, the two groups' average mitral annular systolic velocities (Av. MV Sa) did not differ statistically significantly. (Table 6).

Table (4): Comparison of LV tissue Doppler imaging (TDI) parameters between VSD and control groups

	VSD group (n=30)	Normal group (n=20)	Test value	p-value
Av. MV Sa. (m/s)	0.09 ± 0.03	0.10 ± 0.03	0.614	0.437
Av. MV Ea. (m/s)	0.14 ± 0.01	0.16 ± 0.02	2.916	0.002*
Av.MV E/Ea.	6.88 ± 1.13	6.26 ± 0.97	2.133	0.048*

When comparing the VSD group with the control group in terms of LV 2D strain assessed by speckle-tracking echocardiography (LV-GLS), the VSD group demonstrated significantly lower LV-GLS values (P = 0.02). However, these values remained within the established normal reference limits (Table 7).

Table (5): Comparison of LV 2D strain (LV-GLS) between both groups

	VSD group (n=30)	normal group (n=20)	Test value	p-value
LV-GLS (%)	-20.43 ± 1.92	-21.61 ± 1.27	5.804	0.02*

Subgroup analysis revealed significant higher values of LV dimensions LA dimension & LV volumes. While, there was no significant difference in LV- GLS) in group A compared to their controls (Table 8).

Table (8): Comparison between group A of 2-8 years, and normal group in respect to 2D echo parameters

Parameters	Patient Group (n=14)	Control Group (n=9)	Test value	p-value
LVEDD (mm)	36.14 ± 4.79	31.33 ± 4.18	2.275	0.016*
LA (mm)	27.79 ± 3.49	23.00 ± 2.74	3.067	0.002*
EF %	68.50 ± 5.14	68.89 ± 2.71	0.043	0.837
LV GLS	-21.07 ± 1.85	-22.06 ± 1.32	1.297	0.182

Group B had significantly higher values of LV dimensions, LA dimension & LV volumes. While, significantly lower values of LV-GLS (p = 0.039) compared to the normal group (Table 9). Both age groups maintained normal ejection fractions (p>0.05), highlighting the superior sensitivity of GLS in detecting subclinical dysfunction.

Table (9): Comparison between group B of (9-17 years) and normal group in respect to 2D echo parameters

Parameters	Patient Group (n=16)	Control Group (n=11)	Test value	p-value
LVEDD (mm)	43.44 ± 3.88	39.09 ± 1.58	3.274	0.002*
LA (mm)	32.81 ± 4.31	28.55 ± 1.86	2.674	0.011*
EF %	65.19 ± 4.65	67.64 ± 4.18	1.585	0.174
LV GLS	-19.33 ± 1.85	-21.24 ± 1.16	2.732	0.039*

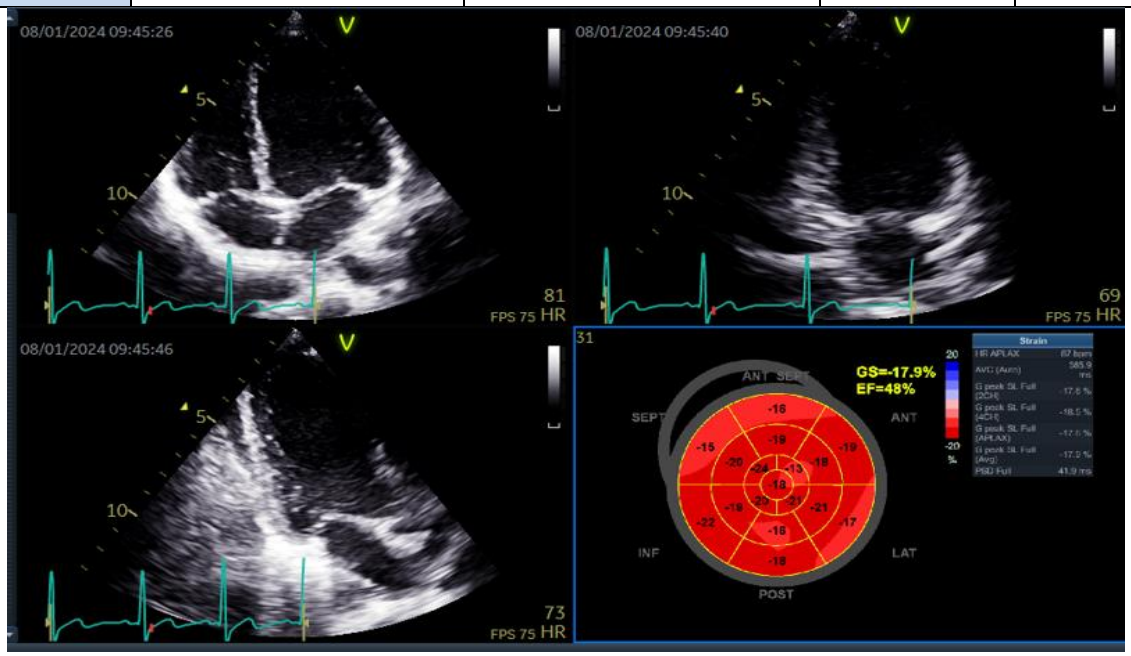

Figure (1): Speckle tracking Echocardiography bull's eye showing decreased GLS (-17.9).

LV-GLS showed strong correlations with various cardiac 2D and echo parameters in VSD patients, highlighting key pathophysiological aspects relationships. The strong negative correlation with age (r=-0.513, p=0.004) suggests progressive myocardial dysfunction over time. While inverse relationships with LV dimensions (LVEDD r=- 0.416, LVESD r=-0.375) and positive correlations with systolic function indices (FS% r=0.444, EF% r=0.460) indicated that ventricular dilation worsens strain. Notably, LV-GLS showed particularly strong associations with biplane EF (r=0.667, p=0.001) and mitral inflow velocities (MV E r=0.531), highlighting its sensitivity to both systolic and diastolic dysfunctions. The negative correlations with VSD characteristics (size r=-0.392, gradient r=-0.466, Qp/Qs r=-0.480) demonstrated that larger shunts cause greater mechanical disadvantage (Table 10).

Table (10): Correlation between LV GLS with different LV echo parameters of the VSD group

Parameters	LV GLS	
	r-value	p-value
LVEDD	-0.416	0.024
LVESD	-0.375	0.041
FS%	0.444	0.014
EF %	0.46	0.011
LA	-0.38	0.029
MV E velocity	0.531	0.003
MV A velocity	0.435	0.016
MV-Ea	0.094	0.623
MV-E/Ea	0.282	0.131

VSD size also correlated positively with the conventional LVEDD reinforcing the link between defect size and cardiac dilation (Table 11).

Table (11): Correlation between LV-GLS with VSD size and shunt parameters

VSD parameters	LV GLS	
	r- value	P- value
VSD size	-0.392	0.017
PG across VSD	-0.466	0.009
Qp /Qs ratio	-0.48	0.007

DISCUSSION

(LVEDD) and left atrial (LA) dimensions were significantly increased in the VSD group compared to the control group, while LVESD, LVEF and fractional shortening (FS) showed no significant differences. These results suggest a state of chronic volume overload and may indicate elevated filling pressures, features commonly associated with incipient diastolic dysfunction. Importantly, these structural changes were strongly correlated with shunt severity (Qp/Qs ratio: 1.63 ± 0.21), particularly in the older age subgroup (9–17 years), who exhibited larger LVEDD and LA dimensions (43.44 ± 3.88 mm and 32.81 ± 4.31 mm respectively; P < 0.05). By contrast, aortic dimension (AoD), LV systolic diameter (LVSD), and LV end-systolic volume (LVESV) demonstrated no significant differences between groups.

These findings match the observations of **Gabriels *et al.*** [16] who reported that even moderate VSDs may result in a significant left-to-right shunt, leading to increased pulmonary blood flow and augmented left-sided filling. Consequently, the increased LV preload contributes to LA and LV dilation.

Similarly, **Ali *et al.*** [17] in a study of 30 children (15 with VSD and 15 controls) demonstrated that the VSD group had significantly higher LVEDD, LVESD and LVEDV compared to controls, both before and after transcatheter closure, highlighting the impact of chronic left-to-right shunting on LV remodeling. Furthermore, **Kotby *et al.*** [18] who studied 30 VSD patients (mean defect size 5.4 mm & predominantly perimembranous) in comparison with 30 healthy controls and reported a significantly higher mean LVEDD and LA dimensions in the VSD group, reinforcing the association between VSD shunt physiology and chamber dilation.

In our study, we demonstrated that there was no significant difference between the patient and control groups regarding EF%, whether assessed by M-mode or 2D echocardiography. This finding is concordant with **Ali *et al.*** [17] who also reported preserved EF in VSD patients compared to controls. Similarly, **Kordon *et al.*** [19] observed that the EF% of the volume-loaded LV can remain within normal limits irrespective of the degree of hemodynamic overload, reflecting preserved systolic function despite chronic shunting. In contrast, **Kotby *et al.*** [18] found that EF was significantly reduced in VSD patients. Nevertheless, EF was preserved in all the studied patients. This discrepancy may be explained by differences in patient characteristics, particularly the size of VSD.

In comparative analysis of TDI parameters of the LV, our study demonstrated that MV-Sa was not significantly different between the VSD and control groups. However, the MV E/Ea ratio was elevated in the VSD group compared to the control group (P = 0.048). This finding, along with the observed increase in LA diameter, suggested that VSD is associated with early structural and functional alterations that can be detected by TDI before overt systolic dysfunction develops. Our results are consistent with **Karonis *et al.*** [20] who evaluated LV function in patients with small, restrictive VSDs and reported that 32 patients (25%) of their cohort, despite having normal LV dimensions, demonstrated LA enlargement suggestive of LV diastolic dysfunction.

Left ventricular (LV) deformation refers to the dynamic changes in ventricular geometry that occur throughout the cardiac cycle, encompassing both systolic contraction and diastolic relaxation. These deformations are inherently three-dimensional, incorporating longitudinal shortening, circumferential contraction, radial thickening and complex torsional mechanics. Accurate assessment of LV deformation is essential, as it provides insight into intrinsic myocardial function and enables the early detection of subclinical dysfunction, often before conventional indices such as ejection fraction are impaired. STE has emerged as a robust technique for quantifying myocardial strain, offering an angle-independent and reproducible evaluation of ventricular mechanics. Importantly, strain imaging has proven valuable in the early detection of myocardial dysfunction in both acquired and congenital heart diseases, including VSD, where chronic volume loading may induce subtle changes in myocardial performance [21]**.** Concordant with the findings of **Adamu *et al.*** [21] the present study demonstrated significantly lower values of LV 2D strain (LV-GLS) in VSD patients compared to control subjects (P = 0.02). However, **Kotby *et al.*** [18] reported no significant difference in LV-GLS between patients and controls. This discrepancy may be attributed to differences in study populations, as their cohort included younger patients with a shorter duration of exposure, potentially limiting the development of detectable subclinical myocardial deformation reflected in strain.

On the other hand, our study included older children who may have had sufficient time for compensatory remodeling. Subgroup analysis in our cohort showed no significant difference in LV-GLS between patients and controls in the younger age group (2–8 years, P = 0.182). Conversely, LV-GLS was significantly reduced in the VSD group aged 9–17 years compared to controls (P = 0.039). This finding suggests the presence of early subclinical ventricular remodeling that is likely related to advancing age and the cumulative effect of long-term left-to-right shunting.

LIMITATIONS

The relatively older age and smaller VSD size of participants, while reflecting an important clinical subgroup, may limit the generalizability of findings to younger patients or those with larger defects, who typically experience greater volume overload. The case–control design permitted detection of associations but did not establish causality, and the absence of longitudinal follow-up restricted assessment of progression or adaptive changes over time. Although comparisons were made with a healthy control group, the lack of serial echocardiographic assessments prevented evaluation of temporal trends in cardiac function. Finally, the modest sample size may limit statistical power and preclude detailed subgroup analyses, particularly regarding the influence of VSD size and type on myocardial mechanics.

CONCLUSION

The results of the present study demonstrated that even small to moderate-sized VSDs can lead to biventricular remodeling and subclinical systolic dysfunction, as evidenced by reduced LV- GLS and right ventricular involvement compared to the healthy control. The strong correlations between VSD size, ventricular dilation, and hemodynamic parameters underscored the importance of advanced echocardiographic techniques, such as speckle-

tracking, for the early detection of myocardial dysfunction.

Funding: NIL.
Conflict of interests: NIL.

REFERENCES

1. **Hoffman J (2013):** Incidence of congenital heart disease: II. Prenatal incidence. Pediatric Cardiology, 34 (3): 417–422.
2. **Penny D and Vick G (2011):** Ventricular septal defect. The Lancet, 377 (9771): 1103–1112.
3. **Van Praagh R, Geva T, Kreutzer J *et al.* (1989):** Ventricular septal defects: How shall we describe, name and classify them? Journal of the American College of Cardiology, 14 (5): 1298–1299.
4. **Lopez L, Houyel L, Colan S *et al.* (2018):** Classification, Epidemiology, and Genetics of Ventricular Septal Defects. Circulation, 137 (12): 618-638.
5. **Rao P, Harris A (2018):** Recent advances in managing septal defects: Ventricular septal defects and atrioventricular septal defects. F1000Research, 7 (1): 498-505.
6. **Minette M, Sahn D (2006):** Ventricular septal defects. Circulation, 114 (20): 2190-7.
7. **Decara J (2012):** Early detection of chemotherapy-related left ventricular dysfunction. Curr Cardiol Rep., 14: 334-341.
8. **Zaca V, Ballo P, Galderisi M *et al.* (2010):** Echocardiography in the assessment of left ventricular longitudinal systolic function: Current methodology and clinical applications. Heart Failure Reviews, 15 (1): 23–37.
9. **Jashari H, Rydberg A, Ibrahimi P *et al.* (2015):** Normal ranges of left ventricular strain in children: a meta-analysis. Cardiovasc Ultrasound, 13: 37.
10. **Chen Q, Cao H, Sun X *et al.* (2014):** The use of chloral hydrate for sedation in children undergoing transthoracic echocardiography.Experimental and Clinical Cardiology, 20(1):1380-1397.
11. **Martin S, Shapiro E, Mukherjee M *et al.* (2014):** Atrial septal defects–clinical manifestations, echo assessment, and intervention. Clinical Medicine Insights: Cardiology, 8: CMC-S15715.
12. **Orsinelli D, Armour A, De Cara J *et al.* (2018):** The American Society of Echocardiography recommendations for cardiac chamber quantification: a quick reference guide from the ASE workflow and lab management task force. J Am Soc Echocardiogr., 28: 1-39.
13. **Nagueh S, Smiseth O, Appleton C *et al.* (2016):** Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. European Journal of Echocardiography, 17 (12): 1321-1360.
14. **Innelli P, Esposito R, Olibet M *et al.* (2009):** The impact of ageing on right ventricular longitudinal function in healthy subjects: a pulsed tissue Doppler study. European Journal of Echocardiography, 10 (4): 491-498.
15. **Levy P, Machefsky A, Sanchez A *et al.* (2016):** Reference Ranges of Left Ventricular Strain Measures by Two-Dimensional Speckle-Tracking Echocardiography in Children: A Systematic Review and Meta-Analysis. Journal of the American Society of Echocardiography: Official publication of the American Society of Echocardiography, 29 (3): 209–225.e6.
16. **Gabriels C, De Backer J, Pasquet A *et al.* (2017):** Long-term outcome of patients with perimembranous ventricular septal defect: results from the Belgian registry on adult congenital heart disease. Cardiology, 136 (3): 147-155.
17. **Ali Y A, Hassan M, El Fiky A *et al.* (2019):** Assessment of left ventricular systolic function after VSD transcatheter device closure using speckle tracking echocardiography. The Egyptian Heart Journal, 71(1): 1.
18. **Kotby A, Abd Al Aziz M, Husseiny *et al.* (2020):** Detection of early myocardial injury in children with ventricular septal defect using cardiac troponin I and two-dimensional speckle tracking echocardiography. Pediatric Cardiology, 41 (8): 1548-1558.
19. **Kordon Z, Rudziński A, Czubaj-Kowal M *et al.* (2002):** The influence of increased preload on left ventricular systolic function in infants with congenital heart disease. Przeglad Lekarski, 59 (9): 728-731.
20. **Karonis T, Scognamiglio G, Babu-Narayan S *et al.* (2016):** Clinical course and potential complications of small ventricular septal defects in adulthood: late development of left ventricular dysfunction justifies lifelong care. International Journal of Cardiology, 208: 102-106.
21. **Adamu U, Schmitz F, Becker M *et al.* (2009):** Advanced speckle tracking echocardiography allowing a three-myocardial layer-specific analysis of deformation parameters. European Journal of Echocardiography, 10 (2): 303-308.